

MARKED UP VERSION OF AMENDED CLAIMS - 0480/001210

4. The process as claimed in claim 1 [one of the preceding claims], where those compounds are read out whose K_i value for binding to 5-HT₅ receptors is also less than 10^{-8} M.
5. The process as claimed in claim 1 [one of the preceding claims], where also at least one 5-HT₅ binding partner-induced action is determined.
7. The process as claimed in claim 5 [or claim 6], where the binding of GTP to G proteins, intracellular calcium levels, the phospholipase C activity and/or the cAMP production are determined.
8. The process as claimed in claim 1 [one of the preceding claims], where, for determining binding affinity and/or activity, the compounds are brought into contact with cellular systems having 5-HT₅ receptors.
14. The use as claimed in claim 11 [one of claims 11 to 13], where the K_i value for binding of the binding partner to 5-HT₅ receptors is less than 10^{-8} M.
15. The use as claimed in claim 1 [one of claims 11 to 14], where the binding partner is a 5-HT₅ agonist.
16. The use as claimed in claim 11 [one of claims 11 to 15], for the treatment of migraine.

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1. An in vitro screening process for the identification of compounds for the treatment of cerebrovascular disorders, which comprises determining the affinity of compounds for 5-HT₅ receptors and reading out those 5-HT₅ binding partners whose binding affinity for 5-HT₅ receptors is greater than their binding affinity for 5-HT_{1D} receptors.
2. The process as claimed in claim 1, where those compounds are read out whose binding affinity for 5-HT₅ receptors is greater by at least the factor 2 than their binding affinity for 5-HT_{1D} receptors.
3. The process as claimed in claim 1, where those compounds are read out whose binding affinity for 5-HT₅ receptors is greater by at least the factor 5 than their binding affinity for 5-HT_{1D} receptors.
4. The process as claimed in claim 1, where those compounds are read out whose K_i value for binding to 5-HT₅ receptors is also less than 10^{-8} M.
5. The process as claimed in claim 1, where also at least one 5-HT₅ binding partner-induced action is determined.
6. The process as claimed in claim 5, where those compounds are read out whose action is agonistic.
7. The process as claimed in claim 5, where the binding of GTP to G proteins, intracellular calcium levels, the phospholipase C activity and/or the cAMP production are determined.
8. The process as claimed in claim 1, where, for determining binding affinity and/or activity, the compounds are brought into contact with cellular systems having 5-

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HTS receptors.

9. The process as claimed in claim 8, where human glioma cell lines or h5-HT5-transfected heterologous cell lines are used.
10. The process as claimed in claim 9, where h5-HT5-transfected CHO cells, h5-HT5-transfected human kidney cells, or h5-HT5-transfected C-6 glioma cells are used.
11. The use of at least one binding partner for 5-HT5 receptors whose binding affinity for 5-HT5 receptors is greater than their binding affinity for 5-HTID receptors, for the production of an agent for the treatment of cerebrovascular disorders.
12. The use as claimed in claim 11, where the binding affinity of the binding partner for 5-HT5 receptors is greater by at least the factor 2 than its binding affinity for 5-HTID receptors.
13. The use as claimed in claim 11, where the binding affinity of the binding partner for 5-HT5 receptors is greater by at least the factor 2 than its binding affinity for 5-HTID receptors.
14. The use as claimed in claim 11 where the K_d value for binding of the binding partner to 5-HT5 receptors is less than 10^{-8} M.
15. The use as claimed in claim 11, where the binding partner is a 5-HT5 agonist.
16. The use as claimed in claim 11, for the treatment of migraine.

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17. The use as claimed in claim 16, for the acute treatment of migraine.